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THE JOURNAL

IT is with considerable pride that those of us who have been associated with this JOURNAL from its beginning, look back over the records of the past two years. The courageousness and ability of the original Board of Editors, in overcoming the seemingly insurmountable obstacles scattered in their path, is still further exemplified by the recollection that those three individuals are now making good in their chosen profession in as many different countries of the world.

We are glad to know that Toronto still retains two of last year's Editorial Board. In a few short months we ourselves will be scattered—who knows where? A glance back through the hurried years causes us to realize that it will not be long before all our present readers will also have entered the ranks of the graduates.

The JOURNAL has reached such a high standard of excellence and has been so encouragingly received that it is with no small degree of trepidation we endeavour to guide its progress through this the third and settled year of its existence.

Modelling itself after the manner of the publications of a number of Old Country Medical Schools, the JOURNAL occupies, we believe, a unique position in this country. It is a regrettable fact that the average undergraduate seldom, if ever, looks at the old and established medical journals. This is only partially compensated for by the knowledge that we, being as yet ill versed in the power of discrimination, thereby escape the error of accepting for the verified truth the multitudinous announcements made in some of these. A function of this JOURNAL, accordingly, is to present to the student, as far as is possible, articles and accounts of recent research of proven worth and modern interest. The material, as a whole, is not readily available in text-books, neither is it an easy source of information for examination purposes. It is only in justice to our senior year and graduate subscribers that each issue contain articles by the staff. Rather than offering another excuse for not deriving benefit from some one or more of the professional journals, we believe this will tend to arouse our interest in such. To the graduate these papers provide a means of keeping in touch with the latest information from his former instructors.

It has always been a policy of the JOURNAL to reprint where deemed advisable; and it is for this reason that, knowing the value of the article, we offer no apology for accepting the courtesies of the Canadian Medical Association Journal in presenting to our readers Dr. Tisdall's paper in this issue.

We hope to hear from many of our graduates. Medicine is a world-wide subject, a fact which we here are apt to overlook. It shall be our policy to strive to broaden the scope of the JOURNAL and, for our own edification as well as in justice to the numerous subscribers giving us support from our sister Canadian Medical Schools, we extend to them a hearty invitation to submit their views and teaching for publication.

In addition to affording a means for closer communication between the Medical Society and the undergraduate body, the JOURNAL has a third mission and it is from this that we derive most benefit. A considerable number of discussion clubs and reading groups are in existence, particularly amongst the upper years. We can speak from personal acquaintance of their value. It is only by actually writing, poring over, revising and rearranging in what we consider the best possible form that we can attain the benefit which invariably accompanies the preparation of our views for publication. We are shortly to go out into the world as educated men and women, and all are certain sooner or later to take some active part in public life. Many of us will be called upon to prepare papers for Association meetings and journals and to defend them during discussion. Our education is far too deficient in such respects. Edinburgh has long been famed for the production of speakers and writers, and credit has always been given to the discussion groups which have been in existence there for years. Such groups cannot be directed by the Faculty. They must be initiated and conducted by ourselves. We sincerely recommend and hope that the junior years will take advantage of their opportunity, only too brief, and start numerous small discussion groups at once. The JOURNAL will be pleased to publish, for the benefit of all of us, the pre-eminent papers presented in such groups. A notable example has been the Seventeen Club which, after an existence of six years, has departed with our graduates of 1925.

An ever present difficulty is in keeping the JOURNAL suitable to such widely divergent interests as those of the first and final years, to take the extremes as an example. This is an obstacle which a graduate publication does not have to contend with. We all, however, have the interests of the profession in common, and the only solution is for an increasing number of contributions from these years of such a nature as to appeal to all readers. It is a significant fact that several of our best contributions in the past have come from junior students.

It is not a policy of the editors to deal with the activities of the alumni. The alumnus thinks of the University as his Alma Mater. He is not sufficiently narrow-minded to be interested only in his old faculty. He always subscribes to and anxiously awaits the coming of the University Monthly. This excellent publication has the whole alumni organization behind it, just as we have our whole faculty, and is much more capable of conducting such affairs than the JOURNAL. We do not feel justified in encroaching upon their domains.

The JOURNAL is the joint property of the undergraduates. We trust that each member of the medical student body will strive to help the publication and himself as well to better attainments by obtaining every issue and, most of all, by writing for it. We of the Editorial Board are deriving untold value and experience from it. We hope that you will become sufficiently interested to share its rewards with us.

A SUGGESTION TO THE PRE-CLINICAL YEARS

IT is now two years since the first issue of this JOURNAL was presented to the student body of the Faculty of Medicine. Many, at that time, were sceptical regarding the purpose of the JOURNAL and doubted if there would be sufficient enthusiasm shown to maintain it for the remainder of the year. As the issues were read by the undergraduates, however, interest grew, and the publication was successful from the beginning. In the second year of its existence, the JOURNAL was fairly generally accepted by virtue of its increasing value, and due to the successful year that preceded it. At first, the greater measure of the interest shown was among those of the senior years, who were engaged in clinical work; but gradually, as articles of interest to the pre-clinical years appeared, they developed interest as well. There were some in the various years who began systematically to collect the issues of the JOURNAL. As each issue appeared articles relevant to the work they were studying were sought for, and read with much interest. Not only did these articles stimulate their interest in the subjects they were studying, but interest developed in the JOURNAL, and they became enthusiastic supporters.

At present those in the junior years of medicine are, we are sure, looking forward to the time when their work will be more largely concerned with patients, not with experimental materials of various laboratories and with text-books. The Editorial Staff desires to encourage interest among the junior years in medicine. We are keen to have the JOURNAL supported, and even to a greater degree than in the past. We believe that the enthusiasm shown by the men of the clinical years for the JOURNAL can be developed in those of the pre-clinical years as well. The younger students are rapidly coming to realize that there are men associated with the Faculty of Medicine whose names are known in almost every locality in which medicine is being taught. We refer to such men as Banting, Macleod, Harding, Klotz, Gallie, and many others; and we of the JOURNAL staff are proud to know that men of this calibre have so generously contributed in the past, and will, we hope, continue to do so from time to time. As an example we make reference to the article by Professor Harding, in this issue, on The Nausea and Vomiting of Pregnancy, with which work he has been so intimately associated.

Interest in the JOURNAL on the part of the junior students in medicine should be stimulated by facts such as these, and additionally by remembering that these men are greatly concerned in Medical Education in the University of Toronto. The aim of this editorial is to encourage interest in the JOURNAL. We believe that this may be aided by pointing out to the students of the junior years the advisability of acquiring articles which will be very valuable to them when they begin to take up clinical work, and perhaps study under men whose names from time to time appear as contributors in the JOURNAL. Therefore our earnest suggestion to the junior medical students is that they begin now to acquire articles which are going to be extremely useful to them during their clinical years in medicine and after graduation as well. The way to do this is to get into possession every issue of the JOURNAL as it comes out, and to keep it for future use, when the experimentation of the laboratory will be followed by the ever interesting study of the patient.

"MYOPIA"

THERE are few undergraduates who do not at some point in their course in the earlier years become disheartened by the seemingly useless detail of innumerable subjects. Many do even seriously question the judgment of those who design the medical curriculum. Yet there seems just one thing to do—accept it all and "muddle on".

The early years are the "stoney age" and one is dealing with the elements of medical science. The first three years in turn seem only to add so much more of that which appears unrelated to the cure of disease. This is the point where, if it were possible, a glimpse at the clinical side of medicine would rekindle enthusiasm and help to overcome the monotony.

For those who suffer from this distortion of vision we promise it is transient, and suggest that they recall their knowledge of the spectrum and remember that the colour elements, many of them vague are all necessary before the pure band of light is possible. This philosophical prescription if thoughtfully applied may establish a new field of vision and perchance may forestall a crisis.

THE NAUSEA AND VOMITING OF PREGNANCY

VICTOR JOHN HARDING

Professor of Pathological Chemistry, University of Toronto

Nausea and vomiting occur in about 50 per cent. of pregnancies. Luckily in the majority of cases it never assumes a serious form and may often be nothing more than an inconvenience. In fact, so widespread has become the belief that a certain amount of sickness accompanies pregnancy, that the phenomenon has become to be regarded as physiological in character, and many physicians neglect entirely its treatment, confident that it will disappear at about the fourth month of pregnancy. Nevertheless, the condition is amenable to therapy, and a case should not be allowed to go on with the symptoms unchecked until it reaches a more severe form.

The symptoms of mild nausea and vomiting of pregnancy have been described often as a "morning sickness". The subject is afflicted with nausea, accompanied with more or less vomiting, on arising. The act of vomiting brings a certain amount of relief, and later in the forenoon the patient recovers and is able to resume her normal habits. The immediate cause of the act of vomiting has been described to the writer as a rising up of fluid or mucus into the œsophagus, and it is the effort to swallow this fluid that brings on a feeling of nausea or the act of vomiting itself. This description, however, may not be true in all cases. Mild cases of nausea and vomiting do not always show symptoms in the early morning. It may occur late in the afternoon, and in this event there will be a repetition of symptoms at the same time of day. This has been called a diurnal or minor periodicity in contrast to the trimestrial or major periodicity, marking its relation to the total pregnancy.

There is no unanimity of opinion that the mild nausea and vomiting of pregnancy is connected with the severer forms, which are usually designated as "pernicious vomiting of pregnancy", or, to give it its title, *hyperemesis gravidarum*. That, however, is the opinion of the writer. The mild cases pass insensibly into cases of a more severe and pernicious type. These are marked by more continuous vomiting; no food may be retained, and even water rejected. Wasting inevitably occurs and the patient may rapidly sink unless relief is promptly and effectually given. Epigastric pain, jaundice, oliguria, and, above all, dehydration, become the more prominent symptoms of the severe case. The end is marked usually by a weak, thready, rapid pulse, followed by collapse.

There have been many theories of etiology proposed, and it must be confessed that even now, although the practice of treatment has greatly improved, we are still in the dark as to the origin of this condition. Sometime ago the writer proposed the theory that either an actual or a relative deficiency of glycogen in the maternal liver might be connected with nausea and vomiting of pregnancy. The idea was based on the fact that the minor periodicity usually followed the period

of longest abstinence from food, and thus partook of the nature of a mild starvation. There is undoubtedly a syndrome which might be called a starvation nausea. Practically no attention has been paid to this; yet it exists, and can be observed in many people; and occasional examples can be found by a close study of the literature. The effect of the starvation might be reinforced by a drain upon the glycogen stores of the maternal liver by the rapidly enlarging foetus and appendages, which would continue until the formation of the placenta offered a buffer between the foetal demands and maternal reserves. Some parts of the theory are undoubtedly open to criticism on quantitative grounds. Nevertheless at the time it offered a definite view-point, looking upon the condition as a phenomenon of starvation, accentuated by pregnancy, and lending itself to a certain amount of experimental and clinical investigation. It would imply treatment by the administration of carbohydrates or glycogen-formers.

There is undoubtedly present in all the more severe cases of vomiting in pregnancy a neurotic element. So much is this evident in some cases that neurosis has been set down as the sole etiological factor by one school of medicine. While the writer would not subscribe to this opinion, the physician treating such a case must never lose sight of this factor. Neglect of this precaution may render void all efforts at treatment.

The treatment of a case of pernicious vomiting of pregnancy admitted to the wards of the Toronto General Hospital follows a definite course. Briefly described, it consists of—isolation, with the administration of sedatives to cover the neurotic factor, and the forcing of fluids in the form of glucose or saline administration to correct the metabolic disturbance. The forcing of fluids as a part of treatment has developed as a result of the intensive study of these cases from the metabolic angle, and it is probable that it is the lack of fluids in the body (dehydration) which represents the difference between the mild case of nausea and vomiting and the pernicious type. The results of observations on case after case have shown that the disappearance of the symptoms on treatment is coincident with the disappearance of the oliguria and the return to a more normal output of urine. *It has been laid down as a standard that the administration of fluids by all routes shall continue until the urine shows a total daily output of* 1 *litre or over, and a specific gravity of 1010 or under, and these results shall be attained within a period of six or seven days.*

The patient, on admission to the hospital, is isolated from the rest of the ward as far as possible. Visitors are forbidden. The bowel is relieved by a simple enema given each morning, and nutrient enemata of 6 ozs. 10% glucose are given t.i.d. and h.s. 30 grains of sod. bromide are given in each enema for the first 24 or 48 hours. No food is given, but fluids are urged by mouth, even if the patient insists that all such fluids are promptly returned. Measurements have shown that usually the output of the vomitus is not greater than the intake of fluid by mouth during the 24 hours, so that some of the fluid is retained and assists in overcoming the dehydration. The amount of fluid given by mouth is set at 60 ozs., although it is rare for the patient to attain this figure the first day or so. The fluids may be varied and consist usually of lemonade, orangeade, water or even weak tea. The patient's taste

is consulted on this matter, and it will be found that it will change, desiring lemonade one day, orangeade or water the next. Any fluid is permissible except strong tea, coffee, cocoa or milk. At the same time 1000 cc. of 5% glucose or normal saline is given daily by the intravenous or interstitial route. This treatment is continued until the urine shows the characteristics italicized in the previous paragraph. In two or three days the patient begins to feel relief and symptoms of hunger appear. Advantage is taken of this, and one or two small light meals are given, consisting of a little toast or cracker, 4 or 5 ozs. of clear soup, and a little stewed fruit. If this is well borne, and it usually is, the meals are increased, and recently we have put the patients directly on a full hospital diet the second or third day after the first appearance of hunger, but giving only half quantities of food. The administration of the daily intravenous or interstitial glucose may be discontinued as soon as the hunger symptoms appear, or the patient definitely shows increased ability to take fluids by mouth. The enemata may be discontinued when the patient is taking and retaining three meals a day, even though these latter be small in amount, but on no account must the oral administration of fluids be discontinued. The urine volume will continue high and its specific gravity low. Whereas on the patient's admission the urine will be found to give a strong test for acetone bodies, these rapidly disappear as the starvation is overcome. Usually the disappearance of the acetone bodies in the urine is coincident with the increased volume and lowered specific gravity of the latter. During convalescenec in hospital and the first two or three weeks after the patient's discharge from hospital the intake of fluids must remain at a high level. It is usual to advise two or three glasses of water or fruit juice between meals.

The administration of fluids thus becomes a more important factor in treatment than the giving of carbohydrate or glycogen-formers. This has been brought about by our observations that only two metabolic factors exist, starvation and dehydration, and of the two dehydration is by far the more prominent in the more serious types. So clearly have we been impressed with the importance of this factor that we strongly suspect it to play a part in the etiology of mild nausea and vomiting of pregnancy, and the treatment of this condition would consist essentially of the giving of fluids rather than a radical change in the diet of the patient. Formerly we advised the giving of meals mainly carbohydrate in nature instead of the usual diet. Such meals would necessarily consist of cereals, fluids and vegetables. Such a regime was found to be successful in the treatment of early nausea and vomiting, and it is possible that the success was due, not so much to the high carbohydrate intake, as to the amount of fluid which necessarily accompanied such a series of meals. Since the inception of the glycogen deficiency theory our knowledge of metabolism has advanced to such a point that we can be sure of a normal glycogen reserve in the liver, provided the food be sufficient in calories and be not overburdened with fat. The patient's ordinary diet, unless it be grossly unbalanced, together with a plentiful intake of water, would thus ensure the conditions of successful treatment of early cases. Such treatment, however, is not so easy as it seems. It requires a great amount of effort on the part of the patient to drink 60 ozs. of fluid.

The coincidence of "diuresis" (high volume and low specific gravity urine) and improvement in clinical condition has been such a constant finding in pernicious vomiting of pregnancy that we have come to look for it with confidence. So much so, that we have taken it as a prognostic sign. The patient whose urine shows these characteristics is not in any clinical danger, even although the vomiting may not have entirely ceased. Very often with such a patient we find at this point a brisk purge to be of value. We have also just recently seen a case where, with the urine volume and specific gravity satisfactory, but the patient continuing vomiting, a still higher intake of fluid resulted in improvement.

All cases, however, do not respond to treatment, and so far it has been impossible to assign a definite cause to failure. Infection undoubtedly plays a part. Even a mild naso-pharyngitis may precipitate another attack of vomiting or render void a properly conducted treatment. Whilst cases of failure of treatment should each be judged on its own merits, it has been found advisable to consider the question of therapeutic abortion should intensive treatment for six or seven days prove unsuccessful. Unless the case has proceeded too far therapeutic abortion undoubtedly offers relief from this condition. The question of therapeutic abortion, however, should be most carefully considered from all angles, and should naturally not be carried out as a hasty measure. Pernicious vomiting, however, if left unchecked, leads to rapid, sudden collapse. A weak, thready pulse may herald such a collapse, but the warning is often short-timed. The adoption of the rule that therapeutic abortion must be seriously considered if six or seven days' treatment does not result in marked clinical improvement, may perhaps err on the side of caution, but it has resulted in the reduction of the mortality from pernicious vomiting to zero in the wards of the Toronto General Hospital.

Quite recently Thalheimer has advocated insulin in pernicious vomiting of pregnancy, basing the use of it on findings in his laboratory of a very much lowered value for plasma bicarbonate, and on a misconception of the theory of etiology proposed by the writer. There can be no doubt of the value of insulin in coma produced by excessive ketosis, and the only question which can arise will therefore be the extent to which cases of pernicious vomiting are liable to coma, or impending coma, from this cause. The records of the laboratory of Pathological Chemistry show only one case in three years where the CO_2 combining power of the plasma gave a figure low enough to be considered as a possible sign of clinical acidosis. All cases, including the one just mentioned, showed a reduction of ketosis on the simple administration of glucose, as described in the previous part of this article. Ketosis in pernicious vomiting of pregnancy is not the same as ketosis in diabetes. It does not arise as a result of inability to burn carbohydrate; it arises from the fact that there is not a sufficient supply of carbohydrate to burn. Given the requisite amount of glucose the ketosis will disappear without the use of insulin. Nor is the ketosis of vomiting of pregnancy as acute as that sometimes found following prolonged anaesthesia; it is of a much milder character, resembling the ketosis of starvation in adults. From this point of view—and we think this to be pertinent—there is no advantage to be gained by the use of insulin.

From chemical examination of the blood, pernicious vomiting of pregnancy has also been quite recently ascribed to a proteose intoxication similar to that of intestinal obstruction, and the use of hypertonic saline advocated. Here again the particular authors have been led astray by superficial observation. It is the fluid given with the salt that brings about improvement in the clinical condition, and the cause is not a lack of sodium chloride. It has been possible to demonstrate this point experimentally.

In conclusion, the writer would like to express his thanks to the Department of Obstetrics which, for the last four years, has placed all cases of pernicious vomiting under the joint observation of its own staff and that of Pathological Chemistry. If any success or improvement in treatment has been attained, or any advance has been made in our knowledge of this subject, it has only been brought about by the loyal co-operation of the two departments.

PARACELSUS*

C. D. S. Leef, '28

Early in the 9th century, A.D., as the result of increasing visits of pilgrims to a tiny shrine on the shores of the Lake of Zurich, the small town of Einsiedeln came into existence. In the course of time a hospital was needed and it became the duty of the abbot, Conrad of Hohenrechsberg, to appoint a physician to take charge. He called Wilhelm von Hohenheim, who had received his degree of Licentiate of Medicine at Lübingen. In 1491 Dr. von Hohenheim married the lady whose position in the hospital corresponded with the matron of to-day, and on November 10, 1493, their son, Theophrastus, was born.

In 1502 Dr. von Hohenheim was appointed town physician at Villach in Karinthia, and Theophrastus accompanied him. While there he attended school at Bleiberg, and the training he received was in analysing the metals and ores obtained from the mines. Later he wrote of this time and mentions working with lead, iron, alum, vitriol, gold, zinc and quicksilver, and of having some knowledge of the chemistry of these substances. In time he went to the famous Benedictine Monastery of St. Andrew's to take his arts degree in order to study medicine at the University of Basel.

It was customary then on entering a university to change one's name to the Latin form. Since there is no Latin for "Hohenheim" Theophrastus was obliged to choose an entirely new name, and he chose "Paracelsus" by which he has since been known.

There was no true science at this time, and at every turn one met the authority of Galen, which was now more than 1200 years old; and it was not even the original Galen, but a version so warped and twisted by numerous translations that the theories of the author were often entirely misrepresented. Paracelsus soon became impatient, and decided that these old dogmas were worthless; so he left Basel and went to Wurtzburg to study with an alchemist named Trithemius. He remained

*First Prize Essay in History of Physiology, 1924-1925.

there for some time, and then, at the age of 22, went to work at the silver mines of Schwatz. Here he studied the chemistry of metals and conceived the idea of using these substances for the curing of disease. At this time he wrote his first book, the *Archidoxa*, in which he tells of these investigations and their results. It is interesting to note that at this time Paracelsus made a salve containing zinc, and from this we have our zinc oxide ointment of to-day.

He stayed at Schwatz for nearly a year and then, as was customary, started to travel. He first visited Vienna, then Cologne, and then Paris, hoping to find some true science, but at every turn he was met by various versions of Galen. He then went south to Montpellier, where the Moorish system of training was used, and again met Galen, this time in the Arabian. Of these old theories he says: "The books of the ancients never satisfied me, for they are not thorough, but serve rather to mislead than to direct." He continued his travels, which included Bologna, Padua, Granada, and Lisbon, and from there sailed to England, where he visited the mines of Cornwall and Cumberland. It is thought that he visited Oxford, but no proof can be found for this. While in England he heard of fighting in the Netherlands, and joined the Dutch army as barber-surgeon, and he tells of his experiences at this time in his book called the *Hospital Book*.

At the conclusion of fighting he rode through Prussia, Bohemia, the Balkan States and Russia to Moscow, and from there to Constantinople. He returned to Italy and served in the Venetian army as surgeon, and was granted a degree by the University of Salerno. He travelled west again, and in 1526 bought citizenship in Strassburg, where he intended to settle.

Meanwhile the Reformation had swept through Switzerland, and authority in Basel was in a chaotic state. The town magistrates were sympathetic to reform, while the University authorities were more conservative; and these were the conditions when, late in 1526, the magistrates of Basel called Paracelsus as town physician. Besides his duties as a physician, this position included a lectureship at the University; and at the very outset Paracelsus was in disfavour with the Faculty because he would not lecture in Latin, but spoke in German. Gradually the rest of the physicians in the city opposed him, partly through jealousy and partly because he would not use Galen as his authority. The apothecaries also opposed him because he mixed his own medicines and referred to their concoctions as "foul broths". He continued to hold his position in the town until at last his opponents planned to remove him by violence, and he was forced to fly for his life. Is it any wonder that at this time of trouble and despair he wrote the words which have been passed down as proof of his excessive egotism: "I tell you the down on my chin knows more than you (the physicians) and all your writers; my shoe-buckles are more learned than Galen and Avicenna."

He travelled back to Alsace and went to Colmar, where he wrote two books, *The Little Surgery* and *On Open and Visible Diseases*. When these were completed he started to travel again, and in 1529 reached St. Gallen. He then journeyed through south and east Germany, and on arriving at Nuremburg, published a book known as *Prognostications*. After leaving Nuremburg his route becomes vague, but in 1536 he was

in Augsberg, and there published a book which he had written since leaving Nuremburg, *The Greater Surgery*.

Many of his older opponents had meanwhile died, and the younger men began to appreciate his work; and these facts, together with the appearance of *The Greater Surgery*, tended to make him more popular among his fellows, and it is known that when he visited Vienna in 1537 he was tendered a banquet by the city officials.

Paracelsus then went to Salzburg, but the privations of these extended journeys had undermined an already delicate constitution, and he felt that he had not much longer to live. On September 21, 1541, he made his will in the presence of six witnesses, whose names are now known, and three days later he died and was buried in St. Sebastian's Cemetery in Salzburg. Later his bones were moved and placed in a niche in an obelisk erected to his memory; and as recently as 1886 these bones were viewed by a German scientist, Dr. Aberle.

It is only in comparatively recent years that anything like a favourable opinion has been held of Paracelsus and his work. Before that time he was regarded as the arch-charlatan and egotist of history. Fuller accuses him of being a drunken quack, and says: "He was not only skilled in natural Magick, but he is charged to converse constantly with familiars. Guilty he was of all vices but wantonnesse." More accurate facts concerning Paracelsus have been found by three German professors—Hartmann, Sudhoff and Aberle—who have investigated some of his original works.

Paracelsus was essentially a chemist or alchemist, and as such tried to explain all the phenomena of life in terms of chemical reactions. In this he was advancing the theories which later came to be held by a group known as the "Iatro-chemists." "Alchemy," he said, "is to make neither gold nor silver; its use is to make the supreme sciences and to direct them against disease." He claimed that the body is composed of three substances—sulphur, mercury and salt. By sulphur he meant volatile matter, by mercury, fluids, and by salt, any substance which was not included in the other two. He thought that in the body were hosts of tiny spirits or archaei, each of which controlled some small part and were in turn controlled by a chief spirit or archaeus. Many of his biographers claim that he believed in astrology and divination, but in his *Paramirum* he himself says: "Stars control nothing in us, suggest nothing, incline to nothing, own nothing; they are free from us and we are free from them."

To the actual advance of medicine Paracelsus did not contribute a great deal. He did know the therapeutic value of sulphur and mercury, and he also used an extract of opium, laudanum. But, as Osler says, one cannot speak very warmly of his medical writings, though one of his books, *The Three Qualifications*, might be accepted to-day as an authority on medical ethics.

Paracelsus added practically nothing to science, yet his contribution must be considered as very great indeed. With the possible exception of Roger Bacon, who preceded him by about 200 years, he was the first man to openly oppose the authority of Galen; and in doing this it was inevitable that he should be regarded as a quack and a charlatan. But

in the light of our modern knowledge it is only right that Paracelsus should be looked upon as one of those who blazed the trail for modern science.

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BLOOD GROUPING AND THE SELECTION OF DONORS FOR TRANSFUSION

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Since the practice of blood transfusion has become a well recognized therapeutic procedure, the matter of the selection of donors for transfusion has come to be one of great importance. The idea of transferring blood from a healthy to an anaemic individual cannot be said to be of recent date, but the putting into practice of this idea was really only developed to any extent during the great war. It was chiefly during this period that various methods of technique were devised by which such a transfer of human blood from one individual to another could be accomplished with any degree of ease.

Unfavourable Reactions

With the wider application of this procedure, however, it was soon noted that while in the majority of cases the patients benefited by the transfusion, in other cases the results were not only disappointing, but occasionally, even disastrous. In some instances the condition of a patient instead of being improved as a result of the transfusion was rendered rapidly and decidedly worse; the outstanding symptoms in these cases being chills, coma, hemoglobinuria and shock. In other instances death would occur almost immediately after the introduction of fresh blood into the circulation of the patient. Occurrences such as this led some observers to conclude that there must be certain individuals whose bloods were incompatible. Previous to this, however, it had been shown that the red corpuscles of certain individuals would undergo agglutination, and later on hemolysis, when brought into contact with blood serum from other individuals. This was at first thought to be a pathological condition associated with and peculiar to certain diseases. Later, however, it was shown [2], [3], [4], that this phenomenon could occur between the cells and serum of perfectly healthy individuals, and the conclusion was reached by different workers, chief among whom were Moss[2] and Jansky,[4] that all human beings fell into one of four groups, depending upon the reaction of their serum to the red cells of other individuals.

Four Blood Groups

These groups were numbered by both workers 1, 2, 3 and 4, but unfortunately their numbers do not represent the same groups in either

case. This has led to a great deal of confusion, since one frequently sees references in the literature to certain blood groups with nothing to indicate which classification is meant. The difference between the two types of classification is best illustrated by the following table:

TABLE I

Comparison of the classifications of Moss and Jansky

Moss Classification		Jansky Classification
Group I	=	Group IV
" II	=	" II
" III	=	" III
" IV	=	" I

For years the Moss classification was largely in use on this continent, but owing to the confusion arising out of the use of the two classifications, the matter was taken up by the American Association of Immunologists and the American Association of Pathologists and Bacteriologists; and, because Jansky's work was published prior to that of Moss, it was decided by these bodies that the classification of Jansky be adopted. Unfortunately, however, many writers, not only on this continent but elsewhere, have not yet fallen in line with this decision, with the result that a great deal of confusion still exists. At the Hospital for Sick Children we have been using Jansky's classification for four years, and that is the classification which will be used throughout this paper.

The distribution of the various groups among the general population seems to be fairly constant in America and Western Europe. In the orient, however, there appears to be a larger proportion of Group III than is found in this country. Our figures, based on grouping several thousand individuals give the proportion in the various groups as follows:

Group	I	42%
"	II	40%
"	III	14%
"	IV	4%

The Agglutination Phenomenon

An attempt to explain this phenomenon of agglutination was made by von Dungern and Hirschfeld[3] in 1911. More recent work along this line using absorption of agglutinin tests, has confirmed the theory of these investigators. Briefly the explanation is as follows:

Certain blood serums contain substances which, when brought into contact with certain red cells, cause these cells to agglutinate. These substances are called agglutinins, they are of two types *a* and *b*. Certain red cells on the other hand possess the property of being agglutinated by these agglutinins. This property is supposed to be due to the presence in the cells of a substance known by the terms agglutinogen, agglutinable body or receptors. We prefer the latter term. These receptors are also of two types and are here designated A & B. Using Jansky's

classification, these agglutinins and receptors are distributed then in the blood groups as follows:

Group	*Cells contain receptors*	*Serum contains agglutinins*
I	..	*a* & *b*
II	A	*b*
III	B	*a*
IV	A & B	..

TABLE 2

Showing the presence in the various groups of receptors and agglutinins.

Agglutination occurs only when corresponding agglutinins and receptors are brought into contact, e.g., agglutinins *a* and receptors A as would be the case were Group III serum mixed with Group II cells. Obviously such a combination does not exist in the blood of any individual, otherwise auto-agglutination would occur.

"The Universal Donor" so-called

The matter then of four blood groups having been established, it was quite generally accepted that the majority of the unfavourable reactions previously reported were caused by the transfusion of incompatible bloods resulting in agglutination and hemolysis. There are those [6], [7], who believe and teach that evil results only follow when the corpuscles of the donor are agglutinated by the serum of the patient; and that while the cells of the patient may be agglutinated by the serum of the donor in the actual test, no ill effects would follow transfusion from such a donor, since the donor's serum is rapidly diluted as it enters the patient's circulation, and does not reach a sufficient concentration to agglutinate his cells. According to this doctrine, then, it should be quite safe to transfuse any individual from a donor whose cells will not be agglutinated by the patient's serum. By reference to Table 2 it will be seen that group I cells contain neither receptors A nor B and, therefore, cannot be agglutinated. For this reason, then, it is claimed that Group I donors can be used irrespective of the group of the patient, and thus to this group has been applied the term "Universal Donor". In my opinion it is most unfortunate that this term has found its way into the literature, since it is most misleading. It gives the impression, as many believe, that it is quite a harmless procedure to transfuse from this group to any other group. This is only partially true, and as partial truth at times is dangerous, it is well to pursue this matter a little further. This I will admit, that accidents following transfusion from Group I to other groups are less likely to occur than if Groups II, III and IV were transfused to other groups; and further I will admit that the dangers of transfusing Group I to other groups in adults are less than they are in children; but since perhaps more transfusions are carried out on children than on adults, and having seen two fatalities in rather close succession, with most intense hemoglobinuria, following a transfusion of Group I to Group II, I am not prepared to accept without reservation the unqualified state-

ment, so frequently seen in the literature, that the blood of Group I may be given individuals in other groups with impunity. It is quite true that both of these cases were children in which, because of the relatively small volume of patient's blood to that of the donor, (using only adults, of course), it is quite an easy matter to introduce the donor's serum in sufficient concentration to cause agglutination and hemolysis of the patient's cells. These accidents occurred about five years ago; and since that time, I have made it a practice to supply only donors in the same group as the patient, and personally never use the term "universal donor" except to draw attention to the fact that, strictly speaking, he does not exist. It is well to remember that Group I Serum contains both *a* and *b* agglutinins, and that every time this so-called universal donor is used for a patient outside his own group, his serum possesses the property of agglutinating the patient's cells; and, if a sufficiently large transfusion is given, the results may be disastrous.

Technique of the Tests

While many methods have been advocated by which the compatibility of bloods may be determined, since this article is intended chiefly for the benefit of the practitioner to whom modern hospital facilities are not available, we shall describe only the simpler methods. For the sake of clearness, let us now rearrange Table 2, placing the serums (together with their agglutinins—the small letters) in the vertical columns, and the cells (together with their receptors—the large letters) in the horizontal rows, thus:

TABLE 3

	SERUM			
Cells	I a & b	II b	III a	IV
I	−	−	−	−
II A	+	−	+	−
III B	+	+	−	−
IV A & B	+	+	+	−

The + sign indicates agglutination.
The − sign indicates no agglutination.

From this table it is seen that whenever corresponding agglutinins and receptors are brought into contact agglutination results: thus, if cells of Group II containing receptors A are brought into contact with serum of Group III, containing agglutinins, *a* agglutination results as shown by the + sign. A careful study of this table, however, will show that in order to determine the group of any one individual it is not necessary to have at hand known sera of all four groups. The

only serum required is a known Group II and a known Group III; and the only information required is that contained in Table 3 in the two columns within the heavy lines, together with the groups of cells at the left hand. These two columns give us four different pictures depending upon the group of cells mixed with these sera. Thus, if no agglutination occurs when the unknown cells are mixed with the known sera, the patient tested is Group I. If agglutination occurs with III and not with II, the patient is Group II. If agglutination occurs with II and not with III the patient is Group III, and if agglutination occurs in both sera, the patient is Group IV.

Assuming then that one has at hand known II and III sera, the technique is carried out as follows:

(1) Mark off a clean glass slide into two divisions with a china pencil, calling the left side II and the right side III; also mark the slide with the name or initials of the individual being tested.

(2) Into division II place a large drop of serum II, and into division III, a large drop of serum III.

(3) Prick the finger or thumb, after cleansing with alcohol and, allowing the skin to dry, and with a very small platinum loop, transfer a small drop of blood (about the size of a pin head) into serum II and mix well.

(4) Flame the loop in a spirit lamp or gas flame, wipe the blood off the finger with a *dry* wipe, and, using a fresh drop of blood, repeat step (3), but in this case transferring the blood to Group III serum.

(5) To prevent evaporation cover the slide with a small inverted dish, and examine at intervals with the naked eye and hand lens, or with the low power of a microscope.

Usually agglutination, if it is going to occur, will be observed in a minute or two. It can be hastened by gently rotating the slide. Since, however, certain cells are agglutinated rather slowly, we have made it a rule not to give a final report until after 20 minutes have elapsed. At the end of that time, by reference to the central portion of Table 3, the group of the individual can be readily determined.

Should there be any doubt as to the grouping, the following procedure known as "cross-agglutination", may be carried out. Obtain from both patient and donor a small amount of serum, by collecting a little blood and centrifuging it, or allowing it to clot. The serum, which must be free from red cells, may be drawn off by a capillary pipette, and a drop transferred to a glass slide as in the original test. To the serum of the donor is then added a small drop of the patient's blood, and to the serum of the patient, a small drop of the donor's blood. If agglutination occurs in either case, that donor should be discarded and another sought.

The above technique is simple, and ordinarily requires only the following outfit; a few glass slides, a needle, a china pencil, a fine platinum loop, a spirit lamp, a hand lens (or a microscope), a little alcohol and a few cotton wipes. In place of the needle can be substituted a fragment of a broken slide, and the platinum loop and spirit lamp can be replaced quite satisfactorily by a few toothpicks.

Preparation of Serum

In addition to the above outfit one will require, of course, a supply of known Group II and Group III serum. When, however, in the course of ones search for donors, individuals in these groups are found, a small amount of serum may be obtained from them and preserved for future use. A common method of preservation is as follows: allow the blood to clot, pipette off the serum and add to each cc. of serum 0.1 cc. of 1% phenol in normal saline. Put the serum into sterile vials, cork and dip the corked ends into melted paraffin. Serum preserved in this manner will keep for many months.

In conclusion we would say that it is not the purpose of this paper to go into the question of the possible existence of sub-groups within the four recognized groups mentioned above. A lengthy discussion of this subject will be found elsewhere.[8]

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NUTRITIONAL DISTURBANCES OF INFANTS AND YOUNG CHILDREN

J. R. Ross, '26

In this paper two of the more frequently encountered nutritional disturbances of infants and young children will be dealt with, chiefly with regard to the dietetic treatment of each, namely, dyspepsia and acute intestinal disease.

DYSPEPSIA

Dyspepsia, sometimes referred to as indigestion, is a nutritional disturbance due to overfeeding with all of the food constituents, especially carbohydrate, and characterized by vomiting, diarrhoea, gas, colic, and usually slight elevation of temperature. It is most common in breast fed babies in the first weeks of life. In these cases it is necessary to find out the baby's nursing gait, or the amount of breast milk obtained at each feeding, so that it can be distinguished from underfeeding. Both conditions have similar symptoms, but in the insufficiently nourished baby the stools are smaller and have no gas and frothiness, the urine excretion is decreased and vomiting comes on later after feeding. With bottle fed babies dyspepsia is most frequent in the summer months and in poor hygienic surroundings.

Causes

1. Too frequent feeding, *i.e.*, less than four hour intervals.
2. Milk sugar and cane sugar are more likely to cause dyspepsia than malt sugar.
3. Condensed milk produces a severe type in young infants.
4. In infants fed top milk mixtures the excessive fat is responsible.
5. In older infants too large amounts of farinaceous foods are frequently the cause of dyspepsia.
6. Attacks of dyspepsia are frequent complications of other gastro-intestinal diseases. These are not considered here.

Pathogenesis

Dyspepsia is a disorder of absorption, metabolism, and to some degree also of digestion. The digestion is usually involved, secondarily, but occasionally primarily. The end products of carbohydrate digestion, which are fermentable acids, produce the stimulation of peristalsis causing the diarrhoea. The acid most frequently found in the stools in large amounts is acetic. These excess acids also stimulate the intestinal mucous glands to secrete excessively, and thus mucous is seen in the stool, which does not indicate an inflammatory process. When abnormal amounts of gas and fermentable acids (lactic, succinic, and acetic) are produced there is a lowering of the carbohydrate tolerance, especially toward the sugar. Normally the sugars ferment rapidly over a small area of gut; the starches, on the other hand, are less laxative because they ferment slowly over a large area of gut and they tend to decrease the diarrhoea. Tundell finds evidence of destruction of body tissue in dyspepsia. The nitrogen retention and fat absorption are lowered and the excretion of Sodium, Calcium and Potassium salts is increased.

Symptoms

1. Vomiting.
2. Eructation of gas.
3. Stools four to six in 24 hours.
4. Distension of the abdomen.
5. Colic.
6. Temperature is slightly above the normal, rarely up to 101° F., and often dipping to 98° F.
7. The urine shows an increased ammonia content but no sugar, albumin or casts.
8. The skin is pale and tissue turgor is decreased.
9. There is often a preceding period of rapid gain in weight, then the weight remains stationary during the dyspepsia or there is distinct loss.

The stools may differ in consistency according to the causative factor. Where sugar is the underlying cause the stool will be green and foamy with curds and mucous present, except where malt sugar is used, producing a brownish stool. Where the fat is the cause of the dyspepsia the stools may be of an oily consistency due to globules of neutral fat; or they may be green, with soap and fatty acid curds.

Prognosis

Dyspepsia in the first three months of life is very often the beginning of a state of marasmus, but in itself is not a fatal condition. It is never necessary to wean a baby because of dyspepsia.

Dietetic Treatment

In the subacute breast fed type

1. Find out the nursing gait.
2. Reduce the feeding from three hour to four hour intervals.
3. One ounce of boiled water before nursing may have to be given.
4. One half ounce of Protein milk may be given before nursing. It has the double advantage of taking the nip off the appetite so that the infant gets less breast milk, and also the presence of the Streptococcus Lacticus tends to reduce fermentation and so relieve the diarrhoea.
5. In the acute type where the child misses some feedings, is drowsy and has no appetite, the treatment varies somewhat according to the severity of the condition. The baby should be put on a starvation diet for the first 12 to 24 hours, only allowing barley water. If the baby is very drowsy glucose solution is preferable. At the end of this period reduced feedings are given, being careful not to overstep the tolerance limit. A careful record of the weight must be kept to see that there is a steady but not too rapid gain. Acute colic is treated with enemata, two teaspoonfuls of glycerine in thirty ounces of water being used, or a soda-mint tablet in one drachm of warm water may relieve the condition. Hot baths are helpful in relieving the colic until the food is regulated.

In bottle fed infants under 3 months

Protein milk is the best food to give if it is properly prepared. After starvation for 24 hours three ounces of protein milk per pound are given in 24 hours. Within one or two days add malt food, beginning with one quarter of an ounce in 24 hours and increasing to one ounce or at most one and one half ounces.

In the more severe cases in infants

1. Starvation for 24 hours; or barley water alone may be given.
2. Skimmed milk and water mixtures may then be given. One ounce of 2% skimmed milk per pound in 24 hours, increasing at the rate of one to two ounces per day till the infant gets one and one half ounces per pound in 24 hours.
3. Gradually replace the skimmed milk with one to two ounces of whole milk at a time.
4. Constantly add cane sugar and malt extract to prevent constipation.

A one to three dilution of whole milk, boiled, with no carbohydrate added may be used in the less severe cases. If fresh milk mixtures are not well borne a cultured Lactic Acid milk is used. Holt recommends its use in a diluted form with equal parts of water. According to Grulee, it is best given undiluted and unsweetened to children over

one year of age. Four feedings of six to eight ounces each are given during the first 24 or 48 hours, other food, such as toast or cereal, being added on the second day.

Dr. Holt advises the use of Protein milk as being one of the most valuable foods in this type of feeding case. He does not use the term dyspepsia, but classes them as difficult feeding cases with loose stools and other gastro-intestinal symptoms predominating. The advantage of this milk is apparently due to its acidity, its low sugar and high protein content. The large amount of protein, which readily undergoes putrefaction, neutralizes by ammonia production the lower fatty acids formed from the fats. For very young infants it is usually used in a diluted form, and it is seldom necessary to reduce the fat by making it from skimmed milk. The easiest method of preparing the food is by using the Protein Milk Powder, but it may also be prepared by adding the curd from whole milk to buttermilk in definite proportions.

The child will not gain much in weight on Protein milk, so carbohydrate should be added as soon as possible, but always with great caution. Dextri-Maltose is the least fermentable of the soluble carbohydrates and should be tried first, cane sugar may then be given, beginning with not more than one half tablespoonful daily. Starchy food may be used with either of these sugars. Give wheat or barley flour one-quarter to one ounce daily, the latter to children five or six months of age. Employed in this way Protein milk may often be continued for two to three months, but without the addition of carbohydrate it cannot be used for more than a few weeks. A change to a diet other than milk should be made very slowly and with great care. Egg yolks or curds are frequently of value in preventing excessive fermentation. These children must be carefully watched until they are three or four years old, to see that they do not have recurring upsets which will lead to a condition of decomposition.

ACUTE INTESTINAL CONDITIONS

Various authors use different nomenclature in describing acute disorders of the intestinal tract in children.

Grulee groups a number of clinical conditions characterized by diarrhoea under the heading "Intoxication." These are Ileocolitis, Summer Diarrhoea, Infectious Diarrhoea and Dysentery. Holt makes a similar grouping under the heading Acute Intestinal Indigestion and Diarrhoea. Kerley also uses the term "Acute Intestinal Indigestion" and includes under it "Acute gastro enteric intoxication" and Acute Colitis (dysentery).

Intoxication

Is an acute affection of the organism characterized by sudden onset with collapse, high fever, diarrhoea, vomiting, leucocytosis and lactosuria, occurring most frequently in the summer months and in artificially fed babies living in poor hygienic surroundings. If acidosis is associated, deep pauseless breathing will be an important sign. Kerley stresses the importance of taking a careful history, which shows that simple diarrhoea probably without vomiting and little fever usually precedes for two or three or more days the more grave acute intestinal indigestion and intoxication. He says there will probably be a larger history of defective intestinal digestion during the previous cold months, and that in only

about 1% of severe gastro-enteric disease of children in summer the onset is sudden, without warning and with urgent symptoms.

Factors Predisposing to Acute Intestinal Conditions

Individual

1. Artificial feeding.
2. Previous alimentary disturbance such as weight disturbance or dyspepsia, or possibly a severe form of decomposition. These prepare for the harbouring of the noxious chemical agent derived from the food or from bacteria.
3. Infection may predispose.

General

1. Heat.
2. Direct action or acute heat intoxication.
3. Chronic action reducing the resistance.
4. Infection of the food, especially milk.

Active

1. Some element of food, *e.g.*, sugar.
2. Decomposition of food.
3. Bacterial infection of the intestinal wall.

Czerney and Keller say that the direct cause is probably the splitting of fats by bacteria into products which are directly irritating to the intestinal canal and which, when absorbed, derange the internal metabolism. They have no direct evidence of these products, but their opinion is supported by animal experiments. The Rockefeller Institute found B. Dysenteriae in a large number of these cases. Other observers found it in only 20% of cases, and say its mere presence is not of etiologic value. On the whole, except in violent epidemics, the bacterial theory of the origin of Summer Diarrhoea has proved very unsatisfactory. Bacteria are probably a more important causative factor of secondary symptoms.

The condition is not only intestinal but metabolic, as shown conclusively by the presence of lactose in the urine during the acute stages. We have, therefore, a severe disturbance of internal metabolism which shows evidence of acidosis, loss of water and salts, and deficiency in the sugar consuming properties.

Acute intestinal conditions may be divided into three classes—Fermentative Diarrhoea, Intoxication, and Infectious Diarrhoea.

I. Fermentative Diarrhoea

This condition constitutes three-quarters of the cases of summer diarrhoea. The symptoms are produced by excessive fermentation. These may again be subdivided, according to degree, into mild, moderately severe and severe.

General Symptoms and Signs

1. Diarrhoea with loss of large quantities of water and salts.
2. Loss of weight.
3. Little, if any, fever. If present it is due to parenteral infection, otitis media, or head cold, etc.

4. Dehydration, loss of tissue turgor, diminished blood flow in the capillaries and a condition of anhydraemia are also associated.

The stools may vary from a loose frothy bubbly green stool to a watery mucous type. They are usually acid in reaction, but may occasionally be alkaline, due to the presence of mucous and dead tissue cells.

Complications

These may be varied; overstepping of the food tolerance is a very serious one. Parenteral infection may also follow.

Treatment

The most important part of treatment is prevention. The morbidity is reduced by education of the parents in the proper feeding of infants. A few of the essentials are:

1. Keep the milk on ice and always boil it before feeding.
2. Clothing must be very light on hot days.
3. Give plenty of cool boiled water.
4. With slight signs or symptoms reduce the diet to one-third or one-quarter of the usual amount, and never force a baby to take its food.
5. In mild cases, where a few loose stools are seen, eliminate the sugar and reduce the amount of food given, but do not reduce the fat.
6. Keep the infant on 2% skimmed milk, give one and one-half ounces per pound of body weight. This will be a relatively high protein diet. One can increase the protein further by adding casein (65% casein) for older children one to three years of age.
7. Vegetables must be entirely eliminated from the diet and the milk must be boiled. A buttermilk-flour formula may be given 180 Cal. X 6. In two or three days buttered rice is added once a day and a half slice of toast. Later allow half an egg yolk hard boiled once a day and a half slice of toast, then a whole egg yolk. Finally allow cereal and a whole slice of toast as the mucous clears up.

Moderately Severe and Severe Types of Fermentative Diarrhoea

Protein milk is the most effective therapeutic agent we have in dealing with these diarrhoeas, because of its high protein content, minimum sugar, moderate fat, and the presence of Lactic Acid Bacillus. Its caloric value is ten to eleven calories per ounce. In the severe types the Protein milk must be diluted because of the greatly decreased tolerance. As soon as there are formed stools, add carbohydrate up to four level teaspoonfuls to the ounce, in the form of starches. If constipation results, change over to soluble carbohydrate, such as corn syrup or cane sugar, but add these cautiously as the constipation is not as serious a condition as the diarrhoea. Fluids are indicated, especially when feeding Protein milk.

The method of treatment suggested by Grulee differs in some respects from the preceding. An outline of the treatment is as follows:

1. (*a*) *Prophylaxis.*—(*a*) Reduce the sugar content to a minimum in hot weather.

(*b*) Reduce the total amount of food in hot weather.

2. *Dietetic Treatment.*—Stop all food. Supply water. Barley water is given in place of the regular bottle every 4 hours. Force fluids between feedings, water may be sweetened with saccharine. Fluids may be

given intraperitoneally, as suggested by McLean and Lang, or they may be given by hypodermoclysis.

Large doses of alkalies are given.

Begin food after 24-48 hours.

Curds of skimmed milk suspended in arrowroot water are sometimes given. Curds from one and one-half drachms of skimmed milk per pound of body weight suspended in arrowroot water, one and one-half tablespoons to the quart of water. Saccharine is used for sweetening. In 24 to 48 hours one-half the skimmed milk curds are replaced by a like amount of whole milk curds, then the mixture is replaced one ounce at a time with skimmed milk and later with whole milk. Carbohydrates are gradually added in the form of malt food and starch, never as milk or cane sugar. The whole period, from the beginning of the starvation diet to the substitution of milk for the curds, should not consume longer than one week. Albumin milk can be used if the buttermilk can be properly prepared. After the period of starvation one ounce of buttermilk per pound diluted with water or barley water is given in four feedings each day for a week. Then it is increased to one and one-half ounces, and gradually replaced by one to two ounces of whole milk with carbohydrates added.

In Infants Over One Year of Age

After starvation give whole milk, one ounce per pound; later one and one-quarter ounces diluted with an equal amount of water. Then add cereals in small quantities. Albumin water, eggs and broths are contraindicated, although vegetable soup may be given.

II. Acute Intestinal Intoxication

This is a more serious disturbance to deal with because there is a toxemia as well as the persistent diarrhoea. There is absorption of split Protein products which are intimately connected with the red blood cells and thus circulated throughout the body. This produces the toxic symptoms. If there is considerable dehydration the poison has its maximum effect. The organs show fatty changes, especially the liver, which undergoes fatty infiltration. In fatal cases there is usually a superficial catarrhal inflammation of the entire gastro-intestinal tract. In cases which recover there is very little, if any, change in the intestinal wall, and it is difficult to find even slight congestion.

Symptoms

The acute intoxication may follow after several days of an apparently mild diarrhoea, especially in the hot weather, and, if improperly treated, some cases develop suddenly.

The infant may be very restless or it may lie in a dull stupor. The skin is hot and dry, the temperature rises rapidly to 102° to 103° F., and sometimes to 106° F. The eyes are sunken and the pulse weak. There may be thirst or complete loss of appetite. Vomiting may be an early and important symptom. Diarrhoea is sometimes delayed for several hours after the beginning of the grave constitutional symptoms. Characteristic features are bursts of flatus following the frequent greenish stools, which have a foul odour. Stools vary from six to twenty per day. After two or three days mucous appears. Microscopical examina-

tion of the stools shows great numbers of epithelial cells, with probably leucocytes and some red blood corpuscles.

Treatment

1. No food is introduced by mouth till all symptoms of intoxication have disappeared.
2. The Toxaemia must first be treated. Dilute or remove the toxic blood by Exsanguination Transfusions. This method has reduced the mortality 50%.
3. Force fluids by nasal drip. Give 5% glucose by mouth, and 10% glucose subcutaneously as a hypodermoclysis. Saline may be given intraperitoneally. It may be necessary to keep the baby nine or ten days without food.
4. When the symptoms of intoxication have gone give food as in Fermentation Diarrhoea. Sugars and fats are not well borne, so Protein milk or Buttermilk are used.

III. Infectious Diarrhoea

This condition is now much less frequently encountered due to improved sanitation and inspection of milk and water supply.

The causes are the Shiga and the Hiss Russell types of the Dysenteriae Bacillus. Different types are found in different communities, and give a variety of lesions. The bacilli produce toxins, and then all cases have a toxaemia as well as the intestinal symptoms. Stools contain blood pus and mucous.

Dietetic Treatment—Holt

The diet in the early stages should be the same as in the forms of acute diarrhoea.

Infants.—For the first 24 to 48 hours barley water or rice water only should be given. As soon as the vomiting ceases Protein milk feeding may be given. At first not more than one or two ounces should be offered every four hours. Force fluids, but do not force the food. Buttermilk may be used as a substitute if Protein milk cannot be obtained, but it is not so efficacious. Avoid all sweet milk mixtures and malted foods, not only during the acute stage, but also during the convalescence. A careful record should be kept of the amount taken in each 24 hours.

Older Children.—Protein milk at first, then junket from which the whey has been carefully strained may also be given with a spoon. At a later period rare scraped beef, buttermilk and egg may be given. Withhold sweet milk, cereals, vegetables and fruits for several weeks if necessary. By this precaution a break in tolerance is avoided.

ENERGY AND FATIGUE

Miss F. M. Quinlan, M.A.

Demonstrator in Physics

The human body is often regarded as a complex machine, in which fuel in the form of food is needed, not only for the performance of work, as in the case of an engine, but also for the repair of the tissues, and

for growth. In order to ascertain the amount of fuel required to maintain the body, it is necessary to determine the amount of energy expended. The energy contained in food, may be measured by means of a bomb calorimeter, which consists of a bomb, in which the food in pellet form is placed, and which is then filled with oxygen under pressure, and placed in a known quantity of water at a known temperature. The pellet is fired by electricity, and the heat produced is taken up by water. The heat of combustion of the food may then be calculated by the general method of calorimetric experiments.

The amount of energy expended may be found by a direct or indirect measurement of the amount of heat lost in the body. The kilogram metre, that is, the work done when one kilogram is lifted through 1 metre, is the unit of work used, although the heat unit—the kilo calorie which is the amount of heat necessary to raise the temperature of 1,000 grams of water 1° Centigrade—is the unit chosen for energy, since eventually all the potential energy of the food consumed or tissues oxidized is reduced to heat, and the external work may be reduced to heat by the mechanical equivalent of heat transformation.

In the direct measurement of the energy lost, the subject is placed in an air-tight, constant temperature chamber; and the change in temperature of the surrounding water is a measure of the heat given off by the subject, after some small corrections have been made. The indirect method consists in the comparison of the expired and inhaled air. The subject of the experiment carries on his back a large bag, which contains two valves, a pipe and a tap. He can breathe in fresh air from the outside atmosphere, and he expels it all automatically into the bag. With a knowledge of the quantity of air, and the composition of the inhaled and expired air, it is possible to determine the oxygen taken in, and the carbon dioxide produced. The decrease of oxygen in the expired air is due to the combustion of the various food-stuffs in the tissues. The amount of oxygen used is multiplied by a factor obtained from the ratio of the amount of carbon dioxide breathed out to the amount of oxygen utilized, giving a statement of the energy assumption in calories. The assumption is made that the amount of oxygen used is a direct index of the amount of material burnt in the tissues. That the assumption is correct is proven by the fact that the two methods give values within one per cent.

Mechanical efficiency, by which we mean the ratio of the external muscle work done to the energy output during the performance, is never greater than 25%. This means that for every 25 foot-pounds of mechanical work done, 75 foot-pounds are degraded into heat inside the body. This value depends also on temperature, relative humidity and other factors. In a maintained muscle contraction, such as holding a heavy object at arms length without moving it up or down, all the energy used is turned into heat, while in running, although part of the energy is turned into kinetic energy, this, in turn, is chiefly reabsorbed into the body as heat.

Experiments on isolated muscles show that a muscle when stretched, like a piece of india rubber, gives out heat. In a single muscle twitch, the rise in temperature is about .003 degrees Centigrade. By means of an accurate thermopile, and a photographic record of the galvanometer response, it is possible to study this heat. It accompanies and is a measure of the chemical processes occurring in muscular activity,

and by following its production continuously it is possible to get the time course of the chemical processes.

Due to the work of W. M. Fletcher, Prof. A. V. Hill and others, it is now known that during the contraction of a muscle, lactic acid is liberated; and when the muscle relaxes, this acid is neutralized by the supply of alkali in the tissues; and after the relaxation is complete, is removed by combination with the oxygen. The system is like an electrical accumulator, which can rapidly perform mechanical work when needed, but must afterwards be slowly recharged by the use of energy to drive the dynamo. So it is possible for a muscle to run up an oxygen debt, which may be paid by the oxygen taken in during the eight or ten minutes following the exercise. Accumulation of lactic acid in the muscle is limited, and when an oxygen debt of 15 litres is reached fatigue sets in. Prof. A. V. Hill has made a study of world's records in various forms of athletics. He draws graphs between average speed and time taken for running and swimming races for men and women. These graphs resemble one another, with a very high speed maintained for a short time, and then the speed diminishing and remaining practically constant after twelve minutes. When the exercise commences, the oxygen intake rises from the low value characteristic of rest, to a high value determined by the value of exercise undertaken. The oxygen used is a measure of the energy expended, one litre of oxygen corresponding to about 5 large calories of energy liberated, or in mechnaical energy, enough to raise one ton 7 feet in the air. This oxygen is not all used up during the exercise, but some may be stored for the oxidation of the lactic acid later.

Curves drawn between oxygen requirement and speed show that the oxygen required per minute increases rapidly with the speed, which means that high speeds and intense efforts are very wasteful. This is due to the fact that in rapid movement, a large proportion of the energy must be used up in overcoming the viscous resistance of the muscles themselves. This waste at high speeds explains why in longer races, the record speeds are much lower than in shorter races, as shown by first graphs mentioned. In anything but the shortest races, an athlete should finish with something near his maximum debt, if he is to do himself justice The maximum effort he can exert over a given interval depends on his maximum oxygen intake, which may be about 4 litres per minute, and his maximum debt. In rowing, maximum efficiency is attained by choosing such a speed that the recovery takes as long as the stroke. The viscous elastic properties of the muscle ultimately determine the length of the oars, and the speed of movement. As an example of the mechanical basis for trainers' precepts, Prof. Hill cites the case of long jumping. In this, success consists in learning to jump high. The record long jump is 25.48 feet. Allowing for the check provided for the vertical impulse, he assumes a horizontal velocity of 30 feet per second. In that case, the performer would remain in the air 0.85 seconds. In that time, using the formula, $s = \frac{1}{2}gt^2$, where t is 0.425, and g is 32, the vertical distance risen would be 2.9 feet. If the man's centre of gravity was originally at a distance of 3 feet from the ground, this corresponds to a high jump of 5.9 feet.

REFERENCE

Prof. A. V. Hill, Presidential Address. Section I. Physiology—British Association Meeting, 1925.

A COMPARATIVE ANALYSIS OF THE SIZE OF CERTAIN PUBLIC INSTITUTIONS IN CANADA, WITH SPECIAL REFERENCE TO MENTAL HOSPITALS

W. C. M. Scott, B.Sc. (Med.), '27

"The most familiar of all public institutions established to administer and foster the general health of the community, and in which charitable effort is manifested, is the general hospital. . . . Second in importance are the houses of refuge and orphanages, homes where destitute adults and homeless children are taken in . . . Asylums for the insane, also found in all the provinces, differ from the foregoing type in that they are in general owned, supported and administered by the province."

Pages 889-890, *Canada Year Book*, 1924.

This statement is typical of the general conception of the relative importance of institutions for the care of the temporarily socially maladjusted but an analysis of the statistics of these various types of institutions discloses data which is quite contrary to this general conception.

For purposes of comparison institutions which care for the socially maladjusted may be grouped into convenient classes in the following manner.

Group I.—General Hospitals
Private Hospitals
Isolation Hospitals
Maternity Hospitals
Sanatoria for Consumptives
Group II.—Mental Hospitals
Group III.—Penitentiaries
Common Gaols
Reformatories for Males
Reformatories for Females
Group IV.—Orphanages
Houses of Refuge

To determine what part each of these groups takes in the care of the total number of socially maladjusted certain facts concerning each group may be compared. The most important comparisons to be made are between: First, the average number of persons in each group of institutions at any given time; second, the number of admittances to each group of institutions per year; and third, the percentage of a year which the average admittee remains in the type of institution to which he is admitted. It is of lesser import to know the number and average size of the institutions within these groups.

During the past summer the writer made an analysis of the statistics of these groups of institutions which he compiled from data furnished by the Dominion Bureau of Statistics. Herein it will suffice to quote the ratios of the average number of persons present in each group of institutions at any time. These ratios for those provinces for which comparative data is available (New Brunswick, Nova Scotia, Quebec and Ontario) show that for every 100 persons present in Mental Hospitals at any given time there are only 73 present in General Hospitals, etc., 38 present in Penitentiaries, etc., and 132 present in Houses of Refuge,

etc. If we compare only the first three groups we are able to add the data for Alberta and British Columbia to the comparison. For every 100 persons in the Mental Hospitals of this large population there are only 83 present in General Hospitals, etc., and 40 in Penitentiaries, etc.

Another way to stress this ratio is to state that the average number of patients in the Mental Hospitals of Eastern Canada is more than 37 per cent. more than those in General Hospitals, etc., and that the number of patients in Mental Hospitals is more than 19 per cent. more than those in General Hospitals, etc., in the provinces of Eastern Canada, Alberta and British Columbia.

This is the type of fact which shows the student that his attitude toward mental patients will be as important in his future practice as his attitude toward any medical or surgical patient. So during his student days he should cease smilingly to relegate the mental patient to a "place of security"—an asylum—but should look upon him as one whose condition is to be investigated with as much effort as that of any other type of patient he meets.

And in the field of mental disease—where the number of patients is increasing and therapeusis is vague—there is as great a stimulus to work with a hope for the future which is based on faith in the power that increasing knowledge brings as in any other field in medicine.

CONNAUGHT LABORATORIES, UNIVERSITY OF TORONTO

DONALD T. FRASER, B.A., M.B., D.P.H.
Assistant Professor of Hygiene and Preventive Medicine

In May 1914, Dr. J. G. FitzGerald, with the consent of the Board of Governors of the University of Toronto, and the sympathetic support of Sir Edmund Osler, established a small department then known as the Antitoxin Laboratory.

In 1915, thanks to the generosity of Colonel Albert Gooderham, of Toronto, a farm of 51 acres (now 70 acres), was secured in York Township, at Fisherville, Ontario, on Dufferin Street, 12 miles from the University. On this property were erected most complete laboratories and stables for the production of public health biological products, diphtheria and tetanus antitoxin, smallpox vaccine, etc. In 1917 this splendid gift, known as the Connaught Laboratories, was formally transferred by Colonel Gooderham to the Board of Governors of the University of Toronto and opened by His Excellency the Duke of Devonshire, then Governor-General of Canada.

According to the terms of the deed of gift, the Connaught Laboratories were donated for the purpose of providing facilities for research in Preventive Medicine and for the production of antitoxins, sera and vaccines of value in public health work.

These laboratories while constituting a separate University Department, are intimately related to the Department of Hygiene and Preventive Medicine of the University. The personnel of the two departments is in large part the same.

The activities of the Connaught Laboratories, for purposes of description, though no sharp line of distinction now exists, may be grouped into Research, Antitoxin, and Insulin Divisions. The latter divisions are not only self-supporting, through the distribution of public health biological products and insulin throughout Canada and elsewhere, but also furnish the major part of the support of the Research Division. Thus, in these laboratories the University of Toronto possesses a somewhat unique organization through which, combined with the Department of Hygiene and Preventive Medicine, scientific investigations of problems in Preventive Medicine, University teaching of Hygiene and Public Health and a public service work of considerable magnitude, are carried on in a co-ordinated fashion to the advantage of each type of activity.

Antitoxin Division

The activities of the Antitoxin Laboratories from their inception in 1914 rapidly increased on account of the urgent demand for biological products for use in the war zone. Immediately enormous quantities of anti-typhoid vaccine, diphtheria antitoxin, antimeningitis serum and tetanus antitoxin were required. During the fall and winter of 1914-15 there was neither time nor opportunity for the Laboratories to supply more than diphtheria antitoxin. The Dominion Government in April 1915 made a grant of $5,000 to equip the laboratory to undertake the preparation of tetanus antitoxin. Before the close of the war more than two hundred thousand packages of tetanus antitoxin were sent overseas; six hundred thousand capillary tubes of smallpox vaccine were supplied to the Canadian Expeditionary Forces. Antimeningitis serum was also supplied and gave very excellent results in the treatment of cerebro-spinal fever amongst the troops. The problem of gas gangrene was also investigated. A potent antiserum was produced against B. welchii, one of the causative organisms of gas gangrene.

One of the aims of the Connaught Laboratories has been to produce the very finest biological products at prices much lower than those charged by commercial laboratories. Many of these products are now supplied to Provincial Depts. of Health throughout Canada. The Department of Health of Ontario and the Provincial Department of Health of Saskatchewan distribute these free of cost.

At the present time the total staff of the Connaught Laboratories in all divisions including the Farm is 63 in number. Of this staff 14 are part time workers. The biological products prepared by the Laboratories are: diphtheria antitoxin, tetanus antitoxin, scarlet fever antitoxin, antimeningitis serum, diphtheria toxoid, toxin for active immunization against scarlet fever, smallpox vaccine, typhoid vaccine, anti-rabies vaccine and Schick and Dick Test material, and insulin.

Insulin Division

The work of the Laboratories has been greatly extended owing to the epoch making discovery of insulin. The co-operation on the part of the Connaught Laboratories was of the greatest value in the development of this extract both for experimental and clinical use. The importance and the volume of this work necessitated the moving of equipment to a separate building, the old Y.M.C.A. Building, for the manufacture of insulin. Insulin from this laboratory is now supplied to all parts of Canada and its distribution has been world-wide.

Research Division

Research in Scarlet Fever

The brilliant researches of Dr. Geo. F. and Dr. Gladys H. Dick of Chicago and of Dr. Dochez of New York in 1923 led the Connaught Laboratories to send the late Dr. Ray Hodge to obtain first hand information from these investigators. Through kindness of the Dicks, Dr. Hodge was given their two strains of Streptococcus haemolyticus scarlatinae with which experimental scarlet fever had been induced in human volunteers. Dr. Dochez also gave him the strain with which he had succeeded in producing a satisfactory curative antiserum. Active pursuit of problems of research in scarlet fever was conducted by Dr. Hodge in the fall of 1924, when his brilliant career was cut short by his death on November the first.

The laboratories now have a large number of horses at the Farm, which are being used for the production of scarlet fever antitoxin. This antitoxin is of great value in the specific treatment of scarlet fever. Its success as a therapeutic agent is more pronounced in those cases where the toxic form of the disease is manifested. However, its efficiency in the rapid production of a temporary immunity in those who have been exposed to scarlet fever, would seem to indicate that we now possess an agent of great importance in the specific control of scarlet fever, comparable in this regard to the prophylactic value of diphtheria antitoxin. Injection of scarlet fever toxin of suitable doses into human beings will produce in them an active immunity against the disease which is apparently of long duration.

One of the serious handicaps in the investigation of scarlet fever is the fact that man is the only suitable animal for the standardization of the toxin and antitoxin. Only such individuals who react to the intracutaneous injection of highly diluted toxin can be used for this purpose. In the past the researchs of the Laboratories in this field have been made possible largely through the co-operation of medical students and nurses. Hence, it is both expedient and fitting that we should appeal to the readers of this Journal to make possible the carrying on of this work by communicating directly or through the class president with Dr. Cowan, Room 53, or to Miss Saunders, Room 26, Medical Building.

The outstanding problems in scarlet fever research are:

1. A simple, rapid and sure laboratory method for the identification of Streptococcus haemolyticus scarlatinae from patients and carriers.
2. The duration of active immunity produced by injections of scarlet fever toxin.
3. A non-toxic antigen which is capable of producing an active immunity.

Research in Diphtheria

Experiments are being carried out to show the efficacy of diphtheria toxoid in producing active immunity, the nature of toxoid and the underlying principles in its production. Toxoid (or anatoxin) is diphtheria toxin made non-toxic by the addition of formaldehyde and applying heat. This new preparation introduced by Ramon of the Pasteur Institute, Paris, may supplant toxin-antitoxin in the active immunization of human beings.

Research in Whooping-cough

Work has just been initiated in an attempt to further the knowledge in regard to whooping-cough.

Research in Tuberculosis

Investigation is being carried out upon certain serological reactions with a view to determining the value of these reactions in relation to the prognosis and diagnosis of tuberculosis in humans. Other researches in this field are in progress.

Research in Vaccinia and other filtrable Viruses

Research is being carried out upon methods of vaccination against smallpox; the nature of the immunity response in revaccinated individuals, and the laboratory diagnosis of smallpox and chickenpox.

Excavations on College Street have been begun for the foundations of the School of Hygiene. This building when completed will house the Department of Hygiene and Preventive Medicine, the Department of Public Health Nursing, and the Connaught Laboratories. Facilities for additional research in the whole field of Preventive Medicine will be provided.

DEFICIENCY DISEASES OF CHILDREN[1]

FREDERICK F. TISDALL, M.D. (Tor.)

The name "deficiency disease" is usually applied to a group of diseases which are caused by a lack in the diet of some one of the various vitamines. In this respect the name is a misnomer. It would be just as rational to consider as deficiency diseases those diseases which are caused by a lack in the diet of any substance. We could quite rightly designate as deficiency diseases the anæmias which are due to a lack of iron, or the abnormalities of the thyroid which result from a lack of iodine; yet these conditions are usually not considered under this heading. By common usage the name is reserved for those conditions which are intimately associated with a deficiency in the diet of any one of the various vitamines. To-day I wish to direct your attention to three of the so-called deficiency diseases which are most frequently encountered in infants and children, namely—rickets, tetany, and scurvy.

Rickets

This is probably more frequently encountered than any other disease of infancy and, I may add, its presence is probably more frequently overlooked than any other condition. The disease occurs in both breast fed and artificially fed infants. It involves not only the osseous system but also other parts of the body, particularly the nervous and

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[1]From the Hospital for Sick Children, Toronto.

Read before the Annual Meeting of the Ontario Medical Society, Toronto, May, 1925.

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muscular systems. The earliest manifestations of the disease are restlessness, head sweating, and tossing of the head from side to side when asleep. Shortly after this the earliest bone changes may be found. Craniotabes or soft areas in the occipital and posterior part of the parietal bones may be felt if firm pressure is used. Beading of the ribs, or the so-called rachitic rosary, which is due to an enlargement of the junction of the bony and cartilaginous portions of the ribs, may be quite readily felt. Later, the other bony changes appear; the enlargement of the epiphyses at the wrists and ankles, the prominent forehead with enlargement of the frontal bosses, the enlargement of the parietal bosses, the flattening of the occipital region of the head, and a general softening of all the bones. The anterior fontanelle is larger than normal, and this combined with the enlargement of the frontal and parietal bosses produces the so-called "hot cross bun" appearance of the head. Fig. 1 illustrates many of these changes. The dentition is delayed. There is marked loss of tone of the muscles and ligaments. The infant does not sit up at the usual age of seven months or stand at the end of the first year. If the infant has been walking it may stop doing so. The abdomen becomes quite large. As a result of the softness of the bones, and the lack of tone of the muscles and ligaments the well known deformities of the chest, spine and extremities may appear.

Not in all infants do the nervous manifestations of the disease appear as prominently as those before mentioned; still they do occur with sufficient frequency to warrant the emphasis which has been placed on them. The most important changes produced in infants by rickets, are not the before-mentioned conditions, but a resultant general lack of resistance to infection. There is no doubt that this general lack of resistance caused by rickets is a very great factor in our infant mortality. The rachitic infants are usually anæmic. They develop head colds, otitis media, bronchitis, and pneumonia more readily than the normal infant, and the outlook is not as favourable. This most important aspect of rickets deserves our careful consideration.

The age and seasonal incidence of rickets is quite definitely defined. The disease may be recognized clinically as early as the second month. The period of greatest incidence however is from the third month to the end of the first year. It may be fairly prevalent in the first half of the second year, but after the second year it is quite unusual to encounter active rickets. In Ontario the disease occurs most frequently in the winter and spring months, from November to May.

The etiology, treatment, and prevention of rickets will be discussed with the next disease that I wish to present for your consideration, namely, tetany.

Tetany

Infantile tetany is encountered fairly frequently at certain seasons of the year. Convulsions are the predominant symptom. In certain cases they may occur as often as thirty or forty times a day. The next symptom, and one which is almost invariably overlooked, is a peculiar inspiratory crow produced when the child cries, and caused by a spasm of the larynx. Some authors consider this laryngeal spasm as a separate disease, but it is only one of the symptoms of tetany. Chvostek's sign, which is a contraction of the facial muscles, and is elicited by tapping the side of the cheek is almost invariably present in infants

with tetany. It is due to a hyperirritability of the facial muscles. This sign is of no significance in infants over two years of age. A characteristic position of the hand (carpopedal spasm) is present in a moderate percentage of cases. Sometimes this position of the hand may be produced by a constriction of the arm for one or two minutes as illustrated in

Figure 1

Fig. 2. When the spasm is produced in this manner it is called Trousseau's sign. The before-mentioned five symptoms of tetany, namely —convulsions, laryngospasm, Chvostek's sign, carpopedal spasm, and Trousseau's sign, are all due to a hyperirritability of the neuro-muscular system.

The age incidence and seasonal incidence of tetany is singularly striking. Of the cases encountered at the Hospital for Sick Children during the past six years 80 per cent. of them occurred at the fifth, sixth, seventh, eighth and nine months of age, and 85 per cent. occurred in the months of January to May inclusive; the highest incidence was

Figure 2

in March and April. It is evident that the age, and seasonal incidence of the disease are indeed most striking.

The blood changes in rickets and tetany are quite definite. It has been shown at the various clinics here and in the States that in rickets the inorganic phosphorus of the blood is reduced, while in tetany it is the calcium of the blood that is reduced. When it is remembered that over 90 per cent. of the inorganic portion of bone is tertiary calcium

phosphate, the reason for the defective formation of bone in rickets is at once evident. In the production of tetany the important factor is the ratio of the sedative to irritating salts in the body. There is in the normal infant a constant ratio between the irritating sodium and potassium salts and the sedative calcium and magnesium salts. In tetany this ratio is disturbed by a reduction of the sedative calcium salt. This results in a hyperirritability of the neuro-muscular system which accounts for all the symptoms of the disease.

The cause of rickets and tetany is intimately associated with a lack of the anti-rachitic vitamine (or anti-rachitic substance) whatever that may be. Why there is in one case a reduction of inorganic phosphorus in the blood, with the production of rickets, and in another a reduction of calcium with the resultant symptoms of tetany, we cannot say. We know, however, that when an adequate supply of this anti-rachitic substance is given, that rickets and tetany will not develop. The interesting discovery has been made in recent years that exposure of the infant to ultra violet rays, which are present in the rays from a mercury vapour quartz lamp or in sunlight, produces the same effect on these inorganic elements in the blood as is produced by giving the anti-rachitic substance. Another observation of the greatest importance has been reported simultaneously in the past few months by Hess of New York and Steenbock of Wisconsin. These investigators showed that the exposure of food containing no anti-rachitic substance to ultra violet rays resulted in the production or formation in the food of the anti-rachitic substance.

It is thus evident that they have actually manufactured one of the so-called vitamines.

The treatment of rickets consists in the administration of the anti-rachitic substance, or exposure of the infant to direct sunlight. The anti-rachitic substance is present in large quantities in cod liver oil that has been suitably prepared. It is the pure oil that has to be given; emulsions are usually of little or no value. In many cases the anti-rachitic content of the average cod liver oil found on the druggist's shelf is very low. We have found in our experience at the Hospital for Sick Children that the best results are obtained by the use of a biologically tested Newfoundland cod liver oil. The dose is one half teaspoonful three times a day, beginning at one to two months of age, and then a teaspoonful three times a day from three months of age on. This amount almost invariably prevents rickets; however, occasional cases are encountered in which it is necessary to increase the dose to get the desired result. As rickets is most prevalent in the winter months it is not possible to expose much more than the infant's face to the direct rays of the sun. There is also an added disadvantage that the ultra violet ray content of the sun's rays during the winter period is very small. Consequently in the winter months dependence should be placed almost entirely on the administration of cod liver oil. In the spring and summer months the skin of the infant should be exposed to direct sunlight. It is to be remembered that glass and clothing cut off all the beneficial rays.

In regard to tetany, the convulsions must be treated. This can be accomplished by the administration of morphin 1/40 to 1/20 grain hypodermically, or the rectal administration of six to eight grains of chloral hydrate or eight to ten grains of sodium bromide. Fifteen to

twenty-five cubic centimetres of a sterile 8 per cent. solution of magnesium sulphate injected subcutaneously is usually quite effective. As the convulsions are a result of the low calcium content of the blood, calcium chloride should be given. The amount should be fifteen grains four or five times a day for the first two days, then the number of doses should be reduced to three a day. This should be continued for three weeks. The calcium chloride may be dissolved in a little water and placed in the feedings. Cod liver oil should be started and continued for a long period, and if possible the infant exposed to the direct rays of the sun.

Scurvy

This occurs most frequently from eight to twelve months of age. Seventy-five per cent. of the cases encountered at the Hospital for Sick Children occurred during this period. It is very rarely seen over one and half years of age. This is of considerable value in the differential diagnosis of scurvy and arthritis of rheumatic origin, as the latter condition is practically unknown under three years of age. The chief symptoms of scurvy are pain on being handled, bleeding and swelling of the gums around the teeth, blood in the urine or stools, and swelling at the ends of the long bones. The swelling is due to a hæmorrhage under the periosteum. This at first glance may appear as a swelling of the joint which often leads to the mistaken diagnosis of arthritis of rheumatic origin. The X-ray appearance is shown in Figure 3. Enlargement of the costo-chondral junctions is also present, but this enlargement is more angular than found with rickets.

Scurvy is caused by a lack of the anti-scorbutic substance. This substance is contained in fresh fruit and vegetable juices. It is practically entirely destroyed by heating. The treatment of scurvy consists in the administration of one-quarter to one-half ounce of orange juice twice a day. The orange juice may be added drop by drop to the cold feeding.

Summary

Rickets is a disease which is very frequently encountered in infants during the winter months. It involves not only the osseous system but other parts of the body, particularly the nervous and muscular systems. The earliest manifestations of the disease are frequently the result of the involvement of the nervous system. The most important result of rickets is a general loss of resistance to infection. There is no doubt that this loss of resistance to infection is a very great factor in our infant mortality during the winter months. The disease can be prevented and eradicated from our province by the routine administration at one month of age of one half teaspoonful of cod liver oil three times a day. This should be increased by the third month to one teaspoonful three times a day. The cod liver oil should contain large quantities of the anti-rachitic substance or vitamine. Many specimens contain only small amounts. As rickets occurs in both breast fed and artificially fed infants the oil should be administered as a routine measure to every infant. As an adjunct to the above treatment the skin of the infant should be exposed, when possible, to the direct rays of the sun.

Tetany is most frequently encountered in infants from five to nine months of age, during the winter and spring months. The symptoms

are: repeated convulsions, spasm of the larynx, hyperirritability of the facial muscles, and carpopedal spasm. The treatment of the disease has been given in detail. The means adopted for the prevention of rickets will prevent the development of tetany.

Scurvy occurs most frequently in infants from eight to twelve months of age. The most prominent symptoms are: pain on being handled, bleeding and swelling of the gums around the teeth, and bleeding from other mucous membranes. The disease can be absolutely prevented by the daily administration to every artificially fed infant of one-quarter

Figure 3

to one-half ounce of orange juice. This treatment should be started at four to five months of age.

Conclusion

In view of the ease with which rickets, tetany, and scurvy can be prevented, the development of any one of these diseases in patients under our care must be regarded as a very serious reflection on our professional ability.

The author desires to acknowledge his thanks to Dr. Alan Brown, Physician-in-Chief, for permission to use the records of the Hospital for Sick Children.

A BIOLOGICAL VIEWPOINT

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For too long a period of time Darwinism and Biology have been synonyms in the popular mind. Darwin attempted to give a rational basis for a developmental series, and arrived at a conception of the survival value of differing characters in a world of struggle and conflict. That specific characters can, save in special cases, have a survival value is now fairly well discredited. As a consequence an attempt to explain specific differences on such a basis is on rather shaky ground. But to the general mind of the world the Darwinian theory stands as the basis of all Biological activity, and when discredited it is supposed to represent the failure of the methods of investigation in Biology. This is a result and an attitude of mind very much to be deplored. Biological science, if it may be defined, is an investigation of the phenomena exhibited by living matter past or present, and is even concerned, as in Pearl's investigations of death, in an attempt at a forecast of future possibilities. That Biology will be able to predict the future in any more certain fashion than a study of past history can give an inkling of what is to come, cannot, of course, be expected.

The problem that Darwin attempted had already been approached by Lamarck, who developed a theory of the change in form of organisms, not based on the observable differences between them, but rather on some internal wish or desire or reaching out of the organism, and an exercise of the parts responsible for its accomplishment. This as a cause of change has little if any experimental or observational support. These two ideas led to an enormous outburst of recorded observations. In the case of Lamarck's thesis the observations are practically all either opposed to the idea or very doubtful. In the case of Darwin's hypothesis the application is certainly much more limited than the first claims made for it. DeVries and Weissmann worked on side lines of the main thesis. DeVries material has by studies in genetics been proven to be the result of an original hybridization and a resultant reappearance of distinct characters in subsequent generations. He worked with characters that find their explanation in the complex nature of the material used. Yet similar studies on other forms, without that disadvantage have led to no real mechanism to explain specific change. Weissmann's concept of a very clear division of the main line of life, the germ-plasm and the recurrent temporary body or soma, in which the former is nourished, and from which it departs, to form new and different bodies at various intervals, gives an idea of the energy exerted by the force of heredity, in going back to first causes and beginning at the beginning; the beginning, at least as far as the microscope can show us, is the single cell. The division is only true partially, and becomes of necessarily less importance as the germ-plasm and the soma become more nearly one and the same thing, as in the lower type of living things. Thus all too briefly put, there is a rather negative aspect developed as to the significance of the phenomena of resemblances that are to be seen in nature. But like all things growing and developing, the truth of biology does not depend on a single theory or on the proof or disproof of ideas that have

arisen. Some of the ideas represent limited truth incapable of explaining more than the phenomena in connection with which they were developed, yet useful as far as they went. It is the scientific aim to keep an open mind on all questions. We must realize that the history of science is a history of partial successes, which are partial usually owing to the limits of human insight, and partial failures which are mainly caused by the effort to spread the ideas gathered from a limited number of observations over the whole complex of recorded phenomena.

If we turn to medicine as a branch of biological science, and begin with an attempt to define it, we are met with great difficulty at the very outset. To one man it means one thing, to another another. Like all things of great value it is difficult to assign a specific value to it. It engages the enthusiasm of the altruist, the reason of the scientist and the compassion of the sympathetic. To one it is the prevention of disease, to another the understanding of diseased conditions, to a third the relief of suffering. It has as many sides as there are types of human beings, and arouses all the forces that determine greatness and pettiness, wisdom and folly in mankind. It never ceases to vary, changing with the spirit of the age, and following the lines laid down by great masters at one time or by temporarily influential men at others. It has great outbursts of advance and curious hiatuses of stagnation. Its view is from the specific to the generalisation as in all true scientific work. So far as its field leads, it produces conclusions that embrace an ever widening number of, at first, apparently isolated facts. The human being and human interactions with other natural phenomena are its chief concerns. It has a specific value as it approaches the real facts of human physical and mental conditions. It tends even in its field to restrict itself to abnormal reactions too much. That is inevitable. The independence of the human type leads to a resentment of interference, a reaction against investigation. It is based, no doubt, on the fear of curiosity, as usually detrimental to the individual rather than beneficial. It is a more terrible thing to be ill than to be well, to be weak than to be strong. Therefore the strong shun the study of weakness in themselves, the well avoid any contact with possible illness for themselves. When actual weakness or illness are present investigation is possible with the hope of improving the situation. In so far as it restricts itself to such methods medicine must give a distorted view of life. It is too localized to certain phases and too intimate with the human point of view to gain the truest perspective.

The biological point of view is general and, as such, tends to the widening of the vision, and gives some concept of the comparative relations of the phenomena in the living world. That in the broadest sense is the relation of biology to medicine. In the attempt to hold this general point of view comparative anatomical work lays too much stress possibly on likenesses, although the essence of comparative study lies in the consideration of both likeness and difference. Yet these series of likenesses are the basis of all classification of all rational arrangement of living matter in anything even remotely resembling order. They are evidences of laws that control life. Similarities in structure, under diverse conditions, give an inkling at least of the type of control that is exerted over protoplasm. Thus we are able to pick out a series of animals, such as amoeba, volvox and hydra, and a series of developmental stages, such as the fertilized egg, the blastula and the gastrula,

and argue from the series parallel that there is some force at work controlling on the one hand the type and on the other the development. The control that gives an earthworm a pronephros; a frog, a mesonephros and a mammal a metanephros, must be the same force that results in the developmental succession in the mammal of pronephric tubules, uniting the coelome and the exterior, (only represented by the female reproductive tube in the adult), of a mesonephros with primitive duct system and, finally, a metanephros with its ducts arising late and uniting secondarily with the original kidney tissue. The development of a series of branchial arches in the embryonic stages of mammals gives some guide as to the forces at work moulding the entire organism. That similar structures are functional in certain lower types shows that the same force that controlled development in one type also controlled it in the other.

This force is ordinarily termed heredity, and if there is one thing more than another, in recent biological studies, that seems reasonably certain, it is the power and control that heredity has over the organism. The fundamental features of living matter depend more upon the kind than upon the circumstances. Development, in individuals, varies with the extent to which circumstances require or allow the hereditary properties to exhibit themselves. Thus Pearl traces the gradual increase, in early years, of the expectation of life from the earliest records to the recent in statistical form. In addition he shows that there has grown up with this a relative lowering of the death rate from many types of diseased conditions. Yet that the hereditary stock has shown very little change is at least strongly indicated by the lowering of the expectation of life as old age is approached. One would argue from that that the inherent properties have remained the same, but conditions surrounding living human beings are improved.

This particular result on the other hand appears to be at least somewhat contradicted by the results of tissue culture. So far as can be determined there is little if any limit to the length of life of isolated cell cultures retained in extremely favourable environmental conditions. The same thing appears to be true of the development of unicellular organisms and of plant cells reproduced indefinitely by budding or cutting. There is then a definite limit to the life of cells associated together in the body. Yet the same cells will live on much longer in separate cultures. In other words there is no use in enlarging the sphere of any idea until we are sure that it applies equally well to the enlarged range of facts.

It would be a very satisfactory thing if we could delude ourselves into the belief that we had arrived at ultimate truth. There would be no more need for struggle and effort to elucidate the infinitely complex phenomena before us. We might rest content. The history of a great many temporarily dominating tendencies in world thought is a history of strife for recognition, followed by triumph, then stagnation and eventually overthrow. This should surely teach us that it is not safe to generalize without limitations. Each idea serves a purpose but must be closely watched, and not permitted to smother with the weight of authority the newer truth that arrives hourly. We are not at the end but at the beginning of our knowledge.

FACULTY NOTES

Bereavements

The Graduating Year wish to extend sympathy and the spirit of friendship to their two class mates who have been lately bereaved of a father. Dr. Fowler of Perth was from our own Alma Mater, and Mr. R. J. Fleming, one of Toronto's leading public men, was very well known, and highly esteemed by all.

Medical Society

The Medical Society Executive announce the appointment of the following committees for 1925-26:

THE MEDICAL AT-HOME COMMITTEE

Honorary Chairman.....Dean A. Primrose
Chairman.....W. L. Deeton
Secretary-Treasurer.....V. B. Laidlaw
Fourth Year Representative.....O. A. Kilpatrick
Third Year Representative.....W. A. McTavish
Second Year Representative.....N. L. Murray
First Year Representative.....B. B. Sparks

TORONTONENSIS COMMITTEE

Sixth Year Representative.....G. C. Kelly
Medical Ladies' Representative....Miss F. M. Smith
Fifth Year Representative.....C. N. Jeffries

INTERFACULTY DEBATING REPRESENTATIVE

V. P. Armstrong, '27

"VARSITY" REPRESENTATIVE

T. H. BELT, '28

Athletic Society

Medicine has certainly shouldered her share in this year's sporting activities.

The year opened with a Medical field day. The Faculty had kindly granted a half holiday, and yet, out of a faculty of seven hundred not fifty showed any interest. It was not a failure, however. The material was good, the best since the Medical field day was instituted, and

Hockey Boots **Skates** **Hockey Sticks**
Sweaters **Pennants** **Squash Rackets**
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competition was keen. Many of the old time and distance records were broken.

100 yards	Lively
220 yards	Johnson
440 yards	Mitchell
880 yards	Mitchell
1 mile	Mitchell
3 miles	Wilson
Shot put	Shute
Discus	Shute
Javelin	Stanton
Pole Vault	Crawford
High Jump	Oakes
Broad Jump	Lively
High Hurdles	Sparrow

Lively and Mitchell were tied for the individual championship. The cup goes to the Third Year. This meet showed its fruit in the Interfaculty Track meet, which was won this year by Meds. for the first time in a great many years. Following this we find that it is left to Meds. to win half of the University's points in the Intercollegiate Track meet. There were six Meds. on that team — Lively, the captain; Mitchell, looked upon as the best one mile runner in Canada; Sparrow, the hurdler; Crawford in the pole vault; Shute in the discus; and Les Black, who came forward when needed and won the javelin throw.

On the University rugby team Meds. are splendidly represented. With the Intercollegiate we find Cliff Weber, the captain. Warren Snyder, an ex-captain is again starring. With these we have Rykert, Long, Dug McFaddyen, Bill Woods Master, and Stringer. All are doing splendid work. To these have been added lately Doc Marritt and Chesh Dreyer from the regular O.R.F.U. squad. We regret that Ken McGibbon met with the unfortunate accident to his knee in the Old Boys' game at the beginning or he would be there as well.

Despite the fact that so many Meds. are playing on the University teams, Senior Meds. are still winning their games as they always have done. Juniors have not been so fortunate.

During November and December there will be an inter-year competition in basketball. Let each year give of its best and some new material may be developed for the Sifton Cup games and also for the University teams.

Sometime in December there will be a Med. Assault-at-Arms. Bear that in mind and turn out. We won the cup last year. We must repeat again. WE CAN DO IT.

Meds. water polo team won a game. Go to it and win more.

Class Pictures

2T6 are having their class pictures for Torontonensis taken at "the Milne Studios." The sittings began on Tuesday, November 10th. Mr. Milne has put aside all other work to devote his whole attention to "Meds."

Graduation Dance

Plans are being made to hold the 2T6 Graduation Dance in December. Speak early for a partner.